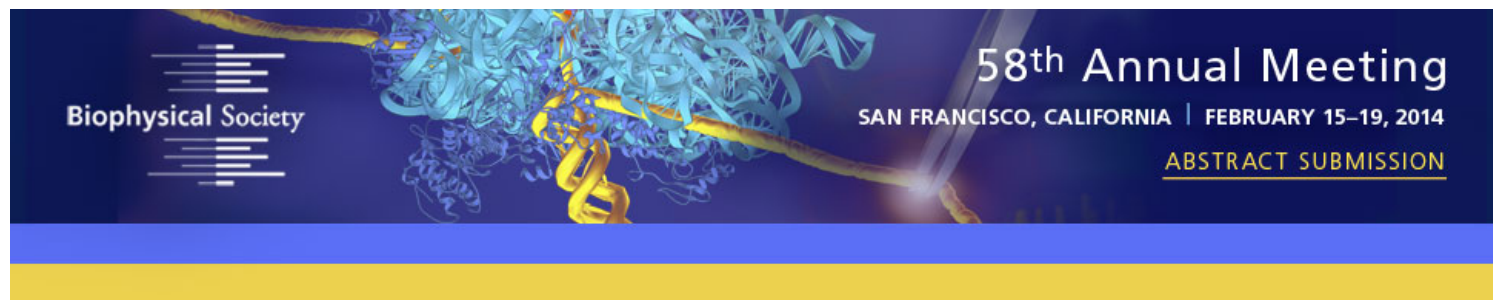




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DETAILED HEMODYNAMIC CHARACTERIZATION OF ATHLETE'S HEART USING LEFT VENTRICULAR PRESSURE-VOLUME ANALYSIS IN A RAT MODEL

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Abstract:

Long-term exercise training is associated with characteristic structural and functional changes of the myocardium, resulting in a condition called athlete's heart. Although exercise-induced left ventricular (LV) hypertrophy has been investigated by several groups in animal models, a detailed hemodynamic characterization is not available. We aimed at understanding the functional and morphological changes in the heart following a three-month-long training period in a rat model.

Athlete's heart in rats was induced by swim training. The swimmer group was exposed to 200 min/day exercise for 12 weeks. The control group swam only 5 min/day.

Following the training period we assessed LV hypertrophy with echocardiography and performed LV pressure-volume (P-V) analysis with a pressure-conductance microcatheter to investigate in vivo cardiac function. Finally, cardiac tissue histology was examined.

Echocardiography showed hypertrophy which was confirmed by LV wall-thickness and heart weight data. Histology also verified LV hypertrophy. We found unaltered heart rate, arterial pressure and LV end-diastolic volume along with decreased LV end-systolic volume, thus increased stroke volume and ejection fraction in the swimmers by invasive hemodynamic measurements. The P-V-loop-derived sensitive, load-independent contractility indexes, such as slope of end-systolic P-V relationship or preload recruitable stroke work were significantly increased. The observed improvement of ventriculoarterial coupling along with increased LV stroke work and mechanical efficiency reflect improved mechanoenergetics of athlete's heart. Despite the significant hypertrophy, we observed unaltered LV stiffness and improved LV active relaxation.

According to our knowledge this is the first study that characterizes functional changes and hemodynamic relations in exercise-induced cardiac hypertrophy.

Conceivably, changes in the active (myosin) and passive (titin) sarcomeric elements underly the described functional myocardial alterations.

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